

CASE REPORTS

• *Methemoglobinemia Simulating Bulbar Poliomyelitis*

• *Hypertrophic Pulmonary Osteoarthropathy*

Methemoglobinemia Simulating Bulbar Poliomyelitis

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A NINE-YEAR-OLD white boy was admitted to the Los Angeles County Hospital January 14, 1951. Several hours earlier he had lost consciousness at school and three physicians who examined him had concurred in a diagnosis of bulbar poliomyelitis.

Two weeks previously the patient had had fever, nausea, vomiting, and diarrhea which lasted four days. Upon physical examination the patient was noted to be acutely ill. The skin was pale and gray. There was pronounced hypoventilation and stupor. The patient vomited yellowish lumpy material which was casually referred to as "eggs" by one observer.

Oxygen under positive pressure was given while tracheotomy was started. The cut tissues and blood were brown, resembling chocolate milk, which suggested the diagnosis of methemoglobinemia. The laboratory reported a methemoglobin concentration of 66 per cent before treatment was instituted. Five milliliters of 1 per cent methylene blue in 45 ml. of normal saline solution was given intravenously. Within an hour the patient was talking, the color of the skin was normal, and the methemoglobin concentration was 22 per cent. The patient then said that he had eaten yellow crayons. (A tooth-marked piece of crayon had been found in a pocket of his garments.) Several hours later the patient was transferred to a private hospital, apparently well except for the tracheotomy incision.

On chemical analysis, the piece of crayon was reported to contain dyes of a group which includes benzidine yellow, vulcan fast yellow, ceylon yellow, and toner yellow. The exact type was not reported because of analytical difficulties on the small quantity of specimen.

Methemoglobin consists partially of iron which has been oxidized from the ferrous to the ferric form that does not transport oxygen. The process is slowly spontaneously reversible and does not damage the red cell. The symptoms result from generalized anoxia.

Treatment consists of the administration of reducing substances. Ascorbic acid acts too slowly and hence is not used in the acute form of the disease. Methylene blue intravenously in doses of 1 to 2 mg. per kg. of body weight given over a five-minute period is safe and effective. Methylene blue acts by speeding the reconversion mechanism of the normal cell.

Primary methemoglobinemia is rare and is due to a congenital biochemical defect in the erythrocytes. Secondary methemoglobinemia is usually due to drugs, of which ni-

trites, sulfonamides, and aniline derivatives are probably the most important. Phenacetin and acetanilid have frequently caused methemoglobinemia owing to their widespread use. Most cases of poisoning due to the ingestion of wax crayons have resulted from orange or yellow crayons. Flinn and co-workers reported that the feeding of wax crayons to animals did not produce methemoglobinemia. They concluded that since relatively few cases of poisoning in humans are reported, considering the frequency of wax crayon ingestion, the occasional poisoning with the material probably can be classed as an idiosyncrasy.

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REFERENCE

Flinn, B., Axelrod, Julius, and Brodie, B.: The toxicity of wax crayons in animals, *J. Ped.*, 33:743, 1948.

Hypertrophic Pulmonary Osteoarthropathy

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THE AUTHORS recently observed a case of hypertrophic pulmonary osteoarthropathy in which the patient was first observed because of massive edema of the legs and the edema was dramatically relieved upon correction of the primary visceral disease.

Hypertrophic pulmonary osteoarthropathy, Marie's disease, was first described independently and almost simultaneously by Pierre Marie and von Bamberger in 1890. Since then, numerous reports have confirmed Marie's original description of the condition as symmetrical periostitis of the four extremities, involving mainly the phalanges and terminal epiphyses of the long bones of the forearm and leg. These changes sometimes involve the bones of the entire limb, and may be associated with dorsal kyphosis and some involvement of the joints, resulting in swelling of soft tissue and tenderness over the involved areas. Subperiosteal calcification may be observed in roentgen studies. The pathologic features are proliferative periostitis with subperiosteal new bone formation.

In the commonly observed simple clubbing of the fingers and toes, the involvement is one of soft tissue proliferation over the terminal phalanges. Osseous change is unusual. When osseous changes of atrophy and absorption of the terminal phalanges do occur, they are a late manifestation. By definition, in hypertrophic pulmonary osteoarthropathy

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